NEURORADIOLOGY
Kinga Karlinger, MD, PhD
Associate Professor
Semmelweis University, Budapest

Diagnostic modalities of the Central Nervous System

- **X-ray: screening is not used any more, x-ray images instead**
  Pneumoencephalography, ventriculography was too invasive, not in use any more.
  Myelography (+CT).
- **Ultrasound (acustic window needed)** neonatal
- **CT:** (x-ray!, c.m.) bone, Ca, air. Soft tissue MDCT, CTA, 3D, dynamic, perfusion examination.
- **MRI** focus on hydrogen ion: MRA, MRS, fMR

Pros: multiplanar, good tissue contrast, without bone artefact, superior imaging of white matter lesions
Cons: bone, Ca, fresh bleeding (stroke differentiation only with advanced sequences) cannot be seen; danger of clips

- **Angiography** /DSA : contrast material needed, use of catheter (invasive)

– mass effect, vascular, vasc. (SAH, AVM) preoperative.
– not for diagnostics, rather for intervention!

- **Nuclear medicine:** SPECT = > PET

– circulation, metabolism

(You should know about MR imaging:
What's in focus (H), the medium (magnetic field), and energy (RF).
Magnets (stable-, electro-: resistive, superconductive)
T1, T2 relaxation, signal localization, flow phenomenon, contrast materials (T1, T2)
Artefacts, hazards, contraindications (absolute, relative) future)

CNS pathology

- **Cerebrovascular:**

Parenchyma (infarction) acute neurological deficit: ischemic (80%), hemorrhagic (15%)
SAH
Hematome: subdural, epidural.

- **Trauma:** fracture (impression! CT), hemorrhage, contusion, diff. diagnostics of subdural vs. epidural bleeding. Shaken baby syndrome

- **Tumor: localization: extra-, intraaxial, supra-, infratentorial, (sellar, parasellar)**

- **Inflammation: bacterial (meninx, parenchyma), viral, parasite (MRI)**

•**Parenchyma lesion: white matter lesions (demyelinisation) MRI**

<u>Head trauma patients' lesions</u>
Primary
Extraaxial hemorrhage
EDH, SDH, SAH,(IVH)
***Intraaxial lesions*:**
Cerebral contusion
Diffuse axonal damage
Deep cerebral and primer brainstem lesion
IVH and choroids plexus hemorrhage

Secondary
Diffuse cerebral oedema
Mass effect with cerebral herniation
Anoxia or hypoxia
Secondary infract with hemorrhage
Infection

•**<u>Cerebral infarction: CT</u>**

•**Hyperacute infarction** (until 12 hours): normal (50-60 %), **hyperdense artery** – Gács sign (25-50 %), the borders of nucleus lentiformis disappear

•**Acute infarction** (12-24 h-ig): ganglia bas. hypodense, insular the border of the cortex-medulla disappears, the sulci compress

•**1-3** day infarction: mass effect, hypodensity (cortex + medulla), possible hemorrhagic transformation (ganglia bas. + cortex)

•**4-7** day: gyri : enhancement, mass effect, persistent oedema

•**1-8** weeks: persistent enhancement, mass effect, transient calcification (mainly infant.)

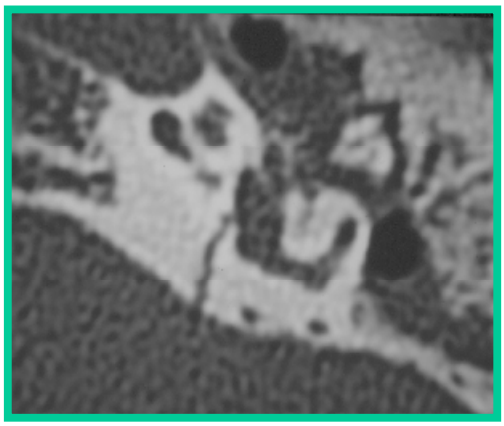
•**Months-years:** encephalomalacy (cyst), parenchyma decrease of the volumen, hydrocephalus localis ex vacuo, (calcification possible)

Most emphasis

- ictus /stroke : ischaemic , haemorrhagic (CT,MR)
- haematoma sub-/epidurale
- columna vertebralis v.s. myelon (CT, MR)

IMAGES:

Os petrosum transv. fractura (laesio n.facialis 50%) (CT):



Contusio cerebri (CT):

Haematoma subdurale:

Haematoma epidurale:

Subacut	ISCHAEM. INFARCT	chronic

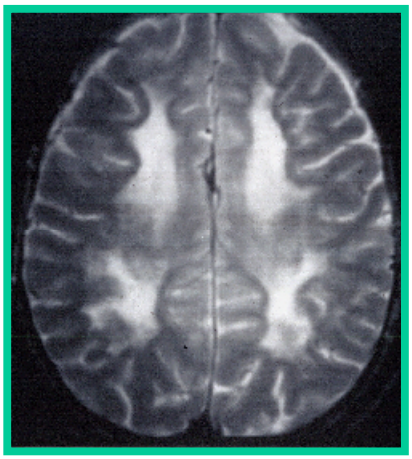
Apoplexia cerebri (CT):

SAH (CT):

Glioblastoma multiforme:

Metatases cerebri:

White matter laesion (MR):

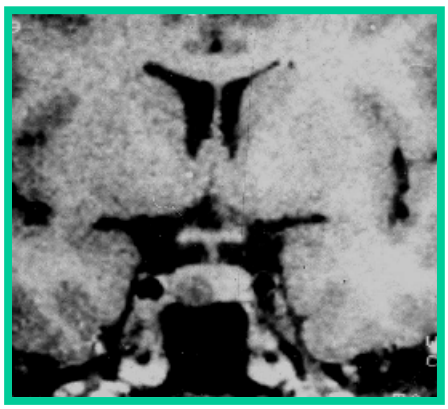


DSA:

MRA:

Intraorbital foreign body (3DCT):

Hypophysis microadenome (MRI):



This document was created with Win2PDF available at http://www.daneprairie.com.
The unregistered version of Win2PDF is for evaluation or non-commercial use only.